

Acquired atheromatous coarctation of the aortic arch

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A 58-year-old woman with a past medical history significant for tobacco use presented with shortness of breath. Physical examination revealed a 30 mm Hg difference in upper-extremity blood pressures (right arm greater than left), elevated jugular venous pressure, and leg edema. A two-dimensional echocardiogram revealed an ejection fraction of 20%. During angiography a heavily calcified lesion was noted in the aortic arch, across which a significant gradient was measured. Computed tomographic scanning identified a focal calcified area in the aortic arch and diffuse atherosclerosis elsewhere. Acquired thromboatheromatous coarctation of the aorta is an uncommon entity found in patients who smoke and are hypertensive. It is almost always seen in conjunction with severe peripheral vascular disease, which this patient had. She was started on heart failure therapy and referred for surgical repair.

A 58-year-old woman with a past medical history significant only for 40-pack-year tobacco use presented with shortness of breath and leg pain with minimal exertion. Physical examination revealed a systolic blood pressure 30 mm Hg greater in the right arm than in the left arm, elevated jugular venous pressure, and bilateral edema of the lower extremities. She had diminished pulses in all four extremities without bruits.

A two-dimensional echocardiogram showed a left ventricular ejection fraction of 20%. The aortic valve was normal. The patient was diagnosed with heart failure and started on a beta-blocker, an angiotensin-converting enzyme inhibitor, and a diuretic. She improved clinically and was referred for coronary arteriography.

Arterial access was obtained via the right common femoral artery. A wire was advanced to the aortic arch, and a calcified lesion was noted in its midportion (*Figure 1*). A pullback pressure measurement across this lesion revealed a pressure gradient of 35 mm Hg (*Figure 2*). The coronary arteries were diffusely calcified, but there was no flow-limiting stenosis. An abdominal aortogram confirmed high-grade stenosis of both iliac arteries.

The patient was referred for computed tomographic angiography (CTA) of the neck, chest, and abdomen as well as Doppler studies of her lower extremities. The CTA showed diffuse atherosclerosis at the origin of all great vessels, including the coronary and carotid arteries. There was a focal calcified stenosis in the aortic arch (*Figure 3*). There was also severe narrowing at the origin of both

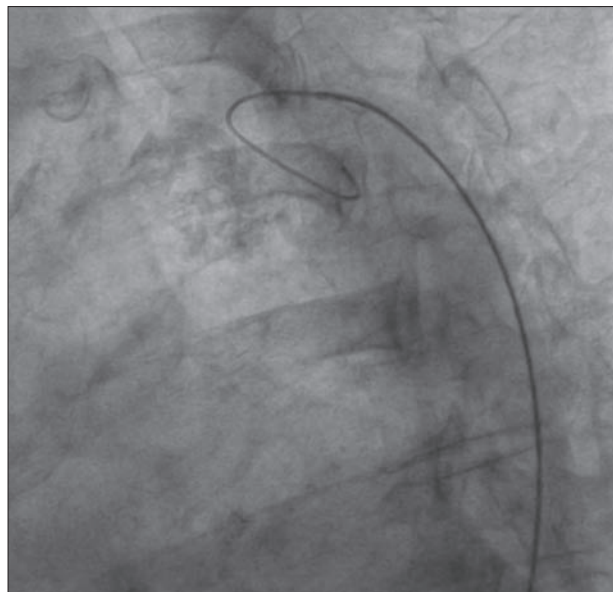


Figure 1. Left anterior oblique radiograph of cardiac catheterization. A J wire is seen abutting a calcified area in the aortic arch.

renal arteries and diffuse atherosclerosis of the aorta. After initiation of medical therapy, echocardiography showed a marked improvement in left ventricular function (40%) and an improvement in diastolic function. The patient was referred for surgical repair of her aortic coarctation.

DISCUSSION

Acquired atheromatous coarctation of the aortic arch is uncommon. It is more frequently found in patients who smoke and are hypertensive. It is almost always seen in conjunction with peripheral arterial disease (1). The majority of information available regarding aortic coarctation is related either to its congenital form or the long-term residual sequelae after its repair. Mention of acquired coarctation secondary to atherosclerosis is relegated to scattered case reports, some of which date back to the 1940s (2).

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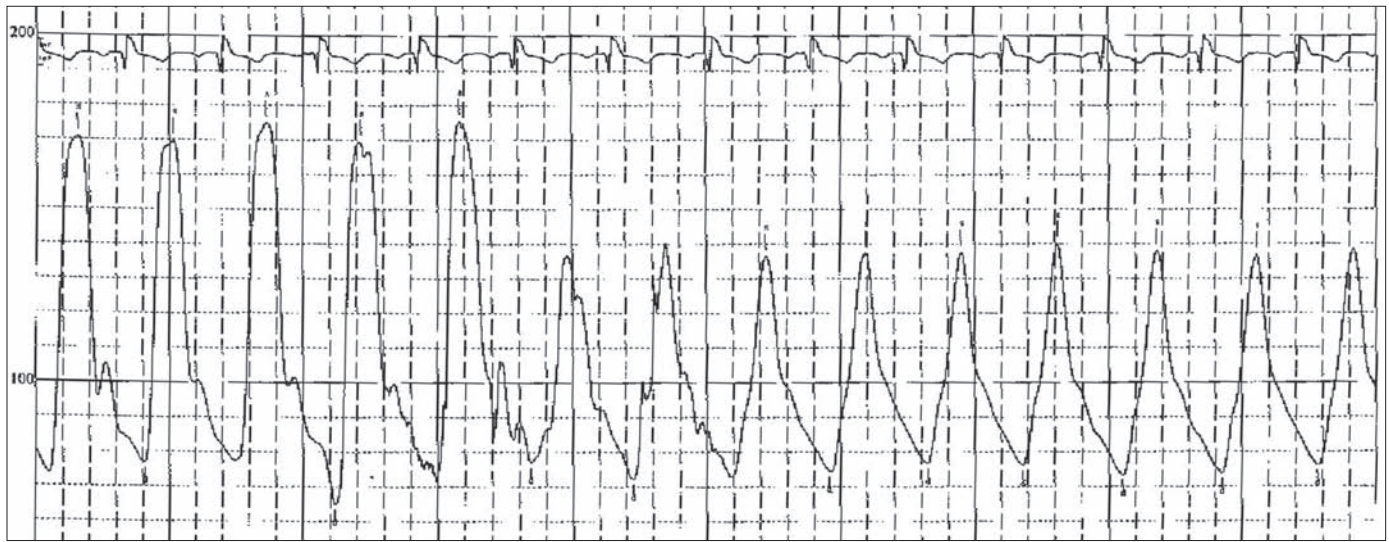


Figure 2. A systolic pressure gradient of 35 mm Hg was measured across the lesion in the aortic arch on catheter pullback.

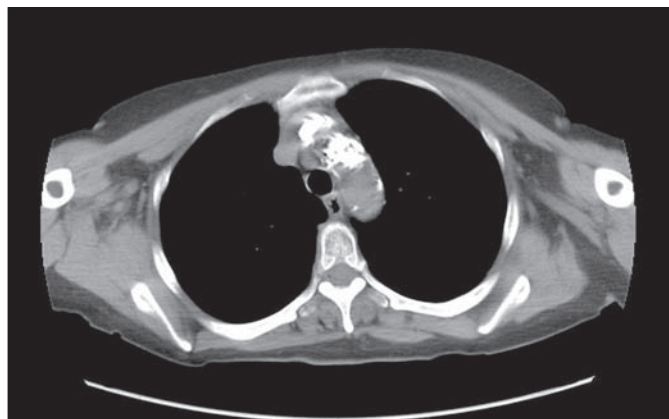


Figure 3. Axial computed tomographic frame showing heavy calcium deposits in the aortic arch.

Atheromatous disease of the aortic arch has a significant risk of embolization. Statins are recommended to reduce this risk, with a low-density lipoprotein cholesterol goal of <70 mg/dL. Oral anticoagulation with warfarin is a consideration for stroke patients with aortic arch atheroma of ≥ 4.0 mm in length. Smoking cessation is recommended (3).

Our case is uncommon in that the patient had no history of coarctation and presented late in life with this finding. The location of stenosis is also atypical since it is proximal to the left subclavian artery. The standard treatment of coarctation of the aorta remains surgical; however, some percutaneous options including balloon dilation and stenting are also available. The experience with these treatment modalities in adults

is limited, with the mean age in most studies not exceeding 30 (4). Our patient was not a candidate for percutaneous intervention due to the extent of calcification present. The most common surgical repair of aortic coarctation is resection with end-to-end anastomosis. In adults, this is complicated by a thicker and calcified aortic wall, as was present in our patient, difficulty mobilizing the affected segments, and the presence of dilated collateral branches. Generally, a prosthetic interposition graft is used to relieve traction. The most common complication following surgical repair is late stenosis and aneurysm. In all cases, poor outcomes are more likely with increasing age (5).

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